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***File 672, TRADEMARKSCAN(R) - Germany
***File 655, TRADEMARKSCAN(R) - Korea
***File 656, TRADEMARKSCAN(R) - Australia

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***File 657, TRADEMARKSCAN(R) - France
***File 673, TRADEMARKSCAN(R) - Italy
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FILES RENAMED
***File 321, PLASPEC now known as Plastic Properties Database
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***File 301, CHEMNAME - please use File 398 ChemSearch
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            $0.00    0.267 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.00  Estimated cost this search
     $0.00  Estimated total session cost   0.267 DialUnits

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? set hi ;set hi
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HILIGHT set on as ''
? b biochem
       06apr09 09:20:26 User226352 Session D1127.2
            $0.00    0.117 DialUnits File410
     $0.00  Estimated cost File410
     $0.02  TELNET
     $0.02  Estimated cost this search
     $0.02  Estimated total session cost   0.384 DialUnits

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         (c) 2009 The HW Wilson Co
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  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
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      Set  Items  Description
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? s transmembrane and domain and (bcma or b cell maturation antigen)
          485220  TRANSMEMBRANE
         2279468  DOMAIN
            1556  BCMA
             124  B CELL MATURATION ANTIGEN
      S1      62  TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION
                  ANTIGEN)
? rd s1
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 2/7/1   (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

0020026412   BIOSIS NO.: 200800073351
cDNA cloning, expression and bioactivity of porcine BAFF
AUTHOR: Guan Zheng-Bing; Dan Wen-Bing; Shui Yan; Ye Ji-Lin; Zhang
  Shuang-Quan (Reprint)
AUTHOR ADDRESS: Nanjing Normal Univ, Life Sci Coll, Jiangsu Prov Key
Lab
  Mol and Med Biotechnol, Nanjing 210097, Peoples R China**Peoples R
China
AUTHOR E-MAIL ADDRESS: zhangshuangquan1201@yahoo.com.cn
JOURNAL: Developmental & Comparative Immunology 31 (12): p1211-1219
2007
2007
ITEM IDENTIFIER: doi:10.1016/j.dci.2007.03.006
ISSN: 0145-305X
DOCUMENT TYPE: Article; Editorial
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: B cell activating factor belonging to the tumor necrosis
factor
  (TNF) family (BAFF) is critical for B cell survival, maturation and
T
  cell activation by acting through its three receptors, BAFF-R, BCMA
  and TACI. In the present study, a porcine BAFF cDNA, designated
pBAFF,
  was cloned by RT-PCR and rapid amplification of cDNA ends (RACE)
  strategies. The full-length cDNA of pBAFF consists of 805 by with a
702
  by open reading frame, encoding 233 amino acids. The deduced amino
acid
  sequence contains a predicted transmembrane domain and a
  putative furin protease cleavage site corresponding to other
identified
  BAFF homologues. The amino acid similarity between the functional
soluble
  parts of pBAFF and human BAFF (hBAFF) or chicken BAFF (cBAFF) is
93% and
  85%, respectively, with identity at the amino acid level was 88%
and 76%,
  respectively. The characteristic of the three-cysteine residues of
BAFF

is conserved in pBAFF. RT-PCR showed that BAFF is expressed in many tissues in the pig, including spleen, liver, lung, heart, intestine, kidney, thymus and PBLs. Recombinant soluble pBAFF (psBAFF) fused with His(6) tag was efficiently expressed in Escherichia coli BL21 (DE3) and its expression was confirmed by sodium dodecyl sulfate polyacrylamide gel electropheresis (SDS-PAGE) and Western blotting. In vitro, purified psBAFF co-stimulates the proliferation of not only porcine B cells but also human B cells. In addition, hsBAFF binds to porcine B cells and has a positive effect on their proliferation. These findings indicate pBAFF plays an important role in proliferation of porcine B cells and functional cross-reactivity occurs between porcine and human BAFF. In vitro expression of bioactive psBAFF provides the basis for further investigation of its potential to be used as an immunoadjuvant for enhancing vaccine efficacy and an immunotherapeutic in pig. It also provides the basis for investigations on the role of BAFF in this important domestic species and an animal model for human diseases.

2/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18631415 BIOSIS NO.: 200510325915
The functional role of DMWD in BAFF-R mediated signal
AUTHOR: Miyazaki Tadaaki (Reprint); Iwai Atsushi; Nakashima Mitsuko; Kimura Chiemi; Uede Toshimitsu
AUTHOR ADDRESS: Inst Genet Med, Kita Ku, Sapporo, Hokkaido 0600815, Japan** Japan
JOURNAL: FASEB Journal 19 (4, Suppl. S, Part 1): pA899 MAR 4 2005 2005
CONFERENCE/MEETING: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences San Diego, CA, USA March 31 -April 06, 2005; 20050331
SPONSOR: Amer Assoc Anatomists
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Amer Soc Pharmacol & Expt Therapeut
Int Union Physiol Sci
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: BAFF (B cell activation factor) is a member of the TNT (tumor necrosis factor)ligand family and binds to three receptors, BCMA (B cell maturation antigen),TACI (transmembrane activator and CAML (Calcium-modulating cyclophilin ligand)-interactor) and BAFF-R (BAFF receptor), mostly expressed on mature B lymphocytes. It has been shown that BAFF has a function for B cell proliferation and maturation primarily through BAFF-R. Here, we identified DMWD (dystrophia myotonica containing WD repeat motif, also called DMR-N9) molecule as a critical signal transducer for BAFF-R mediated signaling. We found that DMWD interacts with the cytoplasmic domain of BAFF-R. However, the function of the DMWD gene products is poorly understood. We confirmed the expression of DMWD in human B cells and found that DMWD has a function to regulate interleukin (IL)-10 production mediated through BAFF-R. Furthermore, we evaluated the function of DMWD for NF-kappa B and JNK activation pathways. It was suggested that DMWD has a critical role for these signaling pathways mediated by BAFF-R. In addition, we identified a kinase as a DMWD-interacting protein and analysed the functions ofthis kinase. We demonstrated that autophosphorylation of this kinase was regulated by DMWD after BAFF stimulation. It was suggested that DMWD regulates thefunction of this kinase to transduce BAFF-R mediated signaling pathways.

2/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18396540 BIOSIS NO.: 200510091040
Identification of proteoglycans as the APRIL-specific binding partners
AUTHOR: Ingold Karine; Zumsteg Adrian; Tardivel Aubry; Huard Bertrand; Steiner Quynh-Giao; Cachero Teresa G; Qiang Fang; Gorelik Leonid; Kalled Susan L; Acha-Orbea Hans; Rennert Paul D; Tschopp Juerg; Schneider Pascal
(Reprint)

AUTHOR ADDRESS: Univ Lausanne, Dept Biochem, CH-1066 Epalinges, Switzerland
**Switzerland
AUTHOR E-MAIL ADDRESS: pascal.schneider@unil.ch
JOURNAL: Journal of Experimental Medicine 201 (9): p1375-1383 MAY 2 05
2005
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: B cell activating factor of the tumor necrosis factor (TNF) family (BAFF) and a proliferationinducing ligand (APRIL) are closely related ligands within the TNF superfamily that play important roles in B lymphocyte biology. Both ligands share two receptors-transmembrane activator and calcium signal-modulating cyclophilin ligand interactor (TACI) and B cell maturation antigen (BCMA)-that are predominantly expressed on B cells. In addition, BAFF specifically binds BAFF receptor, whereas the nature of a postulated APRIL-specific receptor remains elusive. We show that the TNF homology domain of APRIL binds BCMA and TACI, whereas a basic amino acid sequence (QKQKKQ) close to the NH2 terminus of the mature protein is required for binding to the APRIL-specific "receptor." This interactor was identified as negatively charged sulfated glycosaminoglycan side chains of proteoglycans. Although T cell lines bound little APRIL, the ectopic expression of glycosaminoglycan-rich syndecans or glypicans conferred on these cells a high binding capacity that was completely dependent on APRIL's basic sequence. Moreover, syndecan-1- positive plasma cells and proteoglycan-rich nonhematopoietic cells displayed high specific, heparin-sensitive binding to APRIL. Inhibition of BAFF and APRIL, but not BAFF alone, prevented the survival and/or the migration of newly formed plasma cells to the bone marrow. In addition, costimulation of B cell proliferation by APRIL was only effective upon APRIL oligomerization. Therefore, we propose a model whereby APRIL binding to the extracellular matrix or to proteoglycan-positive cells induces APRIL oligomerization, which is the prerequisite for the triggering of TACI- and/or BCMA -mediated activation, migration, or survival signals.

2/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18358730 BIOSIS NO.: 200510053230
Graft-versus-tumor response in patients with multiple myeloma is associated
with antibody response to BCMA, a plasma-cell membrane receptor
AUTHOR: Bellucci Roberto; Alyea Edwin P; Chiaretti Sabina; Wu Catherine J;
Zorn Emmanuel; Weller Edie; Wu Bingyan; Canning Christine; Schlossman
Robert; Munshi Nikhil C; Anderson Kenneth C; Ritz Jerome (Reprint)
AUTHOR ADDRESS: Dana Farber Canc Inst, Dept Med Oncol, 44 Binney St,M530,
Boston, MA 02115 USA**USA
AUTHOR E-MAIL ADDRESS: jeromeritz@dfci.harvard.edu
JOURNAL: Blood 105 (10): p3945-3950 MAY 15 05 2005
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Donor lymphocyte infusions (DLIs) induce effective graft-versus-tumor responses in patients with multiple myeloma who relapse after allogeneic hematopoietic stem-cell transplantation. The graft-versus-myeloma response is presumably mediated primarily by donor T cells, but recent studies have also demonstrated the presence of antibodies specific for a variety of myeloma-associated antigens in patients who achieve complete remission after DLI. One of the B-cell antigens identified in these studies was B-cell maturation antigen (BCMA), a transmembrane receptor of the tumor necrosis factor (TNF) superfamily that is selectively expressed by mature B cells. The present studies were undertaken to characterize the functional significance of antibodies to BCMA in vivo. Using transfected cells expressing BCMA, antibodies in patient serum were found to react with the cell-surface domain of BCMA. Post-DLI patient serum was able to induce complement-mediated lysis and antibody-dependent cellular cytotoxicity (ADCC) of transfected cells and primary myeloma cells expressing BCMA. BCMA antibodies were only found in post-DLI responders and not in other allogeneic transplant patients or healthy donors. These results demonstrate that BCMA is a target of donor B-cell immunity in patients with myeloma who respond to DLI. Antibody responses to cell-surface BCMA may contribute directly to tumor rejection in vivo.

2/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18274167 BIOSIS NO.: 200500181232
Selectivity of BAFF/BLyS and APRIL for binding to the TNF family receptors
BAFFR/BR3 and BCMA
AUTHOR: Day Eric S; Cachero Teresa G; Qian Fang; Sun Yaping; Wen Dingyi;
Pelletier Marc; Hsu Yen-Ming; Whitty Adrian (Reprint)
AUTHOR ADDRESS: Dept Drug DiscoveryCambridge Ctr 14, Biogen Idec Inc, Cambridge, MA, 02142, USA**USA
AUTHOR E-MAIL ADDRESS: adrian.whitty@biogenidec.com
JOURNAL: Biochemistry 44 (6): p1919-1931 February 15, 2005 2005
MEDIUM: print
ISSN: 0006-2960 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: BAFF (B cell activating factor of the TNF family, also known as
BlyS and TALL-I), a TNF family cytokine critical for the development and
function of B cells, has been reported to bind to three receptors, BCMA (B cell maturation protein), TACI (transmembrane activator and CAML (calcium-modulator and cyclophilin ligand) interactor), and BAFFR (BAFF receptor), but with widely conflicting values for the affinity and selectivity of binding. BCMA and TACI additionally bind APRIL (a proliferation-inducing ligand), the TNF family
ligand most homologous to BAFF. Using soluble, monomeric forms of the
receptors, we demonstrate that BAFFR binds BAFF with KD apprx 16 nM, while BCMA binds with KD apprx1.6 muM, indicating a apprx100-fold selectivity for binding to BAFFR over BCMA. APRIL shows the opposite selectivity, binding to BCMA with KD apprx 16 nM while showing no detectable affinity for BAFFR (KD > 3 muM). The binding of
BAFF or APRIL to these receptors is highly sensitive to assay-dependent
avidity effects, likely explaining the widely ranging affinity values
reported in the literature. Binding of BAFF to BCMA-Fc, a bivalent fusion protein consisting of the extracellular domain of BCMA fused to the hinge and CH1 and CH2 domains of human IgG1, in solution or
coated onto an ELISA plate gave apparent binding affinities of apprx0.63
and apprx0.15 nM, respectively, compared to values of KD(app) ltoreq 30

and apprx100 pM for the corresponding BAFFR/IgG1 fusion protein, BAFFR-Fc. The high selectivity of BAFF for BAFFR versus BCMA is thus partly obscured in these multivalent assays. The intrinsically high selectivity inferred from the measurements with monomeric receptor correlates well with in vivo data from knockout mice, providing a possible explanation for the observations that interruption of the BAFFR gene in the A/WySnJ mouse produces a phenotype similar to the BAFF knockout mouse, while the BCMA knockout mouse has no discernible B cell phenotype.

2/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17984372 BIOSIS NO.: 200400355161
Methods and compositions of matter concerning APRIL/G70, BCMA, BLYS/AGP-3 and TACI
AUTHOR: Theill Lars Eyde (Reprint); Yu Gang
AUTHOR ADDRESS: Thousand Oaks, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office
Patents 1285 (2): Aug. 10, 2004 2004
MEDIUM: e-file
PATENT NUMBER: US 6774106 PATENT DATE GRANTED: August 10, 2004 20040810
PATENT CLASSIFICATION: 514-12 PATENT ASSIGNEE: Amgen Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This invention concerns interactions among APRIL/G70, AGP-3/BLYS, BCMA, and TACI and related methods of use and compositions of matter. It has been found that (1) sAPRIL/G70 binds to the cell-surface receptors BCMA and TACI on T and B lymphoma cells, resulting in stimulation of proliferation of primary human and mouse B and T cells both in vitro and in vivo; (2) APRIL competes with AGP3's binding to TACI and BCMA; (3) sBCMA inhibits APRIL and AGP3 binding to its receptors; (4) sBCMA ameliorates T cell dependent and T cell independent humoral immune responses in vivo; (5) sTACI inhibits APRIL and AGP3 binding to its receptors and ameliorates T cell dependent and T cell independent humoral immune responses in vivo; and (6) BCMA exhibits similarity with TACI within a single cysteine rich domain located

N-terminal to a potential transmembrane domain. These discoveries provides a strategy for development of therapeutics for treatment of autoimmune diseases, and cancer, for prevention of transplant rejection. Disease states and disease parameters associated with APRIL and AGP-3 may be affected by modulation of BCMA or TACI; disease states and parameters associated with TACI can be affected by modulation of APRIL; disease states and parameters can be affected by modulation of any of TACI, BCMA, APRIL and AGP-3 by a single therapeutic agent or two or more therapeutic agents together.

2/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17732488 BIOSIS NO.: 200400103245
Chicken BAFF: A highly conserved cytokine that mediates B cell survival.
AUTHOR: Schneider Kirsten; Kothlow Sonja; Schnelder Pascal; Tardivel Aubry; Goebel Thomas; Kaspers Bernd; Staeheli Peter (Reprint)
AUTHOR ADDRESS: Abteilung Virologie, Institut fuer Medizinische Mikrobiologie und Hygiene, University of Freiburg, 79104, Freiburg, Germany**Germany
AUTHOR E-MAIL ADDRESS: staeheli@ukl.uni-freiburg.de
JOURNAL: International Immunology 16 (1): p139-148 January 2004 2004
MEDIUM: print
ISSN: 0953-8178
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Members of the tumor necrosis factor (TNF) family play key roles in the regulation of inflammation, immune responses and tissue homeostasis. Here we describe the identification of the chicken homologue of mammalian B cell activating factor of the TNF family (BAFF/BLyS). By searching a chicken EST database we identified two overlapping cDNA clones that code for the entire open reading frame of chicken BAFF (chBAFF), which contains a predicted transmembrane domain and a putative furin protease cleavage site like its mammalian counterparts. The amino acid identity between soluble chicken and human BAFF is 76%, considerably higher than for most other known cytokines. The chBAFF gene is most strongly expressed in the bursa of Fabricius. Soluble recombinant

chBAFF produced by human 293T cells interacted with the mammalian cell-surface receptors TACI, BCMA and BAFF-R. It bound to chicken B cells, but not to other lymphocytes, and it promoted the survival of splenic chicken B cells in culture. Furthermore, bacterially expressed chBAFF induced the selective expansion of B cells in the spleen and cecal tonsils when administered to young chicks. Our results suggest that like its mammalian counterpart, chBAFF plays an important role in survival and/or proliferation of chicken B cells.

2/7/8 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18203451 Genuine Article#: 338AO Number of References: 24
Title: A novel bioassay for B-cell activating factor (BAFF) based on expression of a BAFF-receptor ectodomain-tumour necrosis factor-related apoptosis-inducing ligand (TRAIL) receptor-2 endodomain fusion receptor in human rhabdomyosarcoma cells
Author(s): McClements M; Williams S; Ball C; Bristow A; Wadhwa M; Meager A (REPRINT)
Corporate Source: Natl Inst Biol Stand & Controls,Blanche Lane, Cytokine & Growth Factor Sect, Biotherapeut Grp,S Mimms EN6 3QG/Herts/England/ (REPRINT); Natl Inst Biol Stand & Controls,Blanche Lane, Cytokine & Growth Factor Sect, Biotherapeut Grp,S Mimms EN6 3QG/Herts/England/; Natl Inst Biol Stand & Controls,Blanche Lane, Prot Sci Sect, Technol Dev & Infrastruct Grp,S Mimms EN6 3QG/Herts/England/
Journal: JOURNAL OF IMMUNOLOGICAL METHODS, 2008, V337, N1 (AUG 20), P63-70
ISSN: 0022-1759 Publication date: 20080820
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS
Language: English Document Type: ARTICLE
Abstract: B-cell activating factor (BAFF) is a type II transmembrane glycoprotein belonging to the tumour necrosis factor ligand superfamily. Active soluble forms of BAFF are generated either by cleavage of the extracellular domain or by recombinant DNA technology. The current bioassay for measuring the activity of soluble BAFF involves stimulation of the proliferation of mouse splenic B-cells

in the presence of goat anti-mouse IgM mu chain which is rather cumbersome and lengthy and yields variable results. We have therefore developed an alternative functional assay which relies on the ability of BAFF to induce an apoptotic response in human rhabdomyosarcoma cells. For this, we constructed a chimeric receptor containing the ectodomain of the MuBAFF-R - the major cell receptor for BAFF - and the endodomain of the HuTRAIL-R2 - one of the two functional receptors for TRAIL - which is known to contain a death domain and trigger apoptosis. When the chimeric receptor was expressed in the TRAIL-sensitive human rhabdomyosarcoma cell line KD4 clone 21, recombinant BAFF of either human OF mouse sequence stimulated apoptosis, similar to TRAIL, in a dose-dependent manner. The transfected cell population, called FL17, expressing the MuBAFF-R/ HuTRAIL-R2 thus provided the basis of a novel functional bioassay for BAFF that is simple and relatively fast to perform. The construction of the chimeric receptor, development of the transfected cells expressing this receptor and the development of sensitive and reproducible bioassays for BAFF and anti-BAFF neutralising antibodies are described.

2/7/9 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09831500 Genuine Article#: 453UW Number of References: 20
Title: The role of TALL-1 and APRIL in immune regulation
Author(s): Khare SD (REPRINT) ; Hsu HL
Corporate Source: Amgen Inc,Dept Pathol Pharmacol,1 Amgen Ctr Dr/Thousand Oaks//CA/91320 (REPRINT); Amgen Inc,Dept Pathol Pharmacol,Thousand Oaks//CA/91320; Amgen Inc,Dept Inflammat,Thousand Oaks//CA/91320
Journal: TRENDS IN IMMUNOLOGY, 2001, V22, N2 (FEB), P61-63
ISSN: 1471-4906 Publication date: 20010200
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND
Language: English Document Type: EDITORIAL MATERIAL
Abstract: Members of the tumor necrosis factor (TNF) superfamily play important roles in cell proliferation and death during immune regulation. Most members are synthesized as type II transmembrane proteins; the carboxy terminal extracellular domain can be cleaved from the cell membrane to form soluble active cytokines that bind to appropriate members of the TNF receptor family Here, we

describe the biological significance of recently discovered members of the TNF superfamily (TALL-1 and APRIL) and their receptors (TACI and BCMA) in the pathophysiology of human diseases.

2/7/10 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07240275 Genuine Article#: 140DX Number of References: 59
Title: The characterization of murine BCMA gene defines it as a new member of the tumor necrosis factor receptor superfamily
Author(s): Madry C; Laabi Y; Callebaut I; Roussel J; Hatzoglou A; LeConiat M; Mornon JP; Berger R; Tsapis A (REPRINT)
Corporate Source: UNIV PARIS 11,INSERM CJF 95 02, CTR RECH, FAC MED PARIS SUD, 32 RUE CARNETS/F-92140 CLAMART//FRANCE/ (REPRINT); UNIV PARIS 11,INSERM CJF 95 02, CTR RECH, FAC MED PARIS SUD/F-92140 CLAMART//FRANCE/; UNIV PARIS 06,CNRS UMR C7590, LAB MINERAL CRISTALLOG/F-75005 PARIS//FRANCE/; INST GENET MOL,INSERM U301/F-75010 PARIS//FRANCE/
Journal: INTERNATIONAL IMMUNOLOGY, 1998, V10, N11 (NOV), P1693-1702
ISSN: 0953-8178 Publication date: 19981100
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND
Language: English Document Type: ARTICLE
Abstract: The BCMA gene is a new gene discovered by the molecular analysis of a t(4;16) translocation, characteristic of a human T cell lymphoma. It has no significant similarity with any known protein or motif, so that its function was unknown. This report describes the cloning of murine BCMA cDNA and its genomic counterpart. The mouse gene is organized into three exons, like the human gene, and lies in murine chromosome 16, in the 16B3 band, the counterpart of the human chromosome 16p13 band, where the human gene lies. Murine BCMA cDNA encodes a 185 amino acids protein (184 residues for the human), has a potential central transmembrane segment like the human protein and is 62% identical to it. The murine BCMA mRNA is found mainly in lymphoid tissues, as is human BCMA mRNA, Alignment of the murine and human BCMA protein sequences revealed a conserved motif of six cysteines in the N-terminal part, which strongly suggests that the BCMA protein belongs to the tumor necrosis factor receptor (TNFR) superfamily, Human BCMA is the first member of

the TNFR family to be implicated in a chromosomal translocation.

2/7/11 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

0005585389 SUPPLIER NUMBER: 2004081899
TWE-PRIL; a fusion protein of TWEAK and APRIL
Kolfschoten G.M.; Pradet-Balade B.; Hahne M.; Medema J.P.
AUTHOR EMAIL: j.p.medema@lumc.nl
CORRESP. AUTHOR/AFFIL: Medema J.P., Department of Clinical Oncology, Leiden
University Medical Center, Albinusdreef 2a, 2333 ZA Leiden, Netherlands
CORRESP. AUTHOR EMAIL: j.p.medema@lumc.nl
Journal: Biochemical Pharmacology (Biochem. Pharmacol.), v66, n8, (1427-1432), 2003, United States
PUBLICATION DATE: October 15, 2003 (20031015)
CODEN: BCPCA
ISSN: 0006-2952 eISSN: 1471-2970
DOI: http://dx.doi.org/10.1016/S0006-2952(03)00493-3
PUBLISHER ITEM IDENTIFIER: S0006295203004933
RECORD TYPE: Abstract; New
DOCUMENT TYPE: Conference Paper
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 61

TWEAK and APRIL are both members of the tumor necrosis factor family, which are involved in respectively angiogensis and immune regulation. While TWEAK is processed at the cell surface, APRIL is processed inside the cell by a furin-convertase and is solely able to perform its function as a soluble factor. Recently, TWE-PRIL has been identified, which is an endogenous hybrid transcript between TWEAK and APRIL. TWE-PRIL is a transmembrane protein that consists of a TWEAK intracellular, transmembrane and stalk region combined with APRIL as its receptor-binding domain. As such TWE-PRIL is expressed at the cell surface. Although TWE-PRIL, like APRIL, can stimulate T and B cell lines, distinct biological functions that may result from its membrane anchoring cannot be excluded. Understanding the function of this newly identified protein will contribute to the elucidation of the complexity of the tumor necrosis factor family.

2/7/12 (Item 1 from file: 72)
DIALOG(R)File 72:EMBASE

0082845246 EMBASE No: 2009045695
Physiological roles and mechanisms of signaling by TRAF2 and TRAF5
ISSUE TITLE: TNF Receptor Associated Factors (TRAFs)
Au P.-Y.B.; Yeh W.-C.
University Health Network, Department of Medical Biophysics, University of Toronto, Toranto, ON, Canada
AUTHOR EMAIL: wyeh@uhnres.utoronto.ca
CORRESP. AUTHOR/AFFIL: Yeh W.-C.: University Health Network, Department of Medical Biophysics, University of Toronto, Toranto, ON M5G 2C1, Canada
CORRESP. AUTHOR EMAIL: wyeh@uhnres.utoronto.ca
EDITOR(S): Wu H.
Weill Medical College of Cornell Uni, New York, NY, United States

Advances in Experimental Medicine and Biology (Adv. Exp. Med. Biol.) (United States) December 1, 2007, 597/- (32-47)
CODEN: AEMBA ISSN: 0065-2598 ISBN: 9780387706290
DOI: 10.1007/978-0-387-70630-6-3
DOCUMENT TYPE: Book Series; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 126
TRAF2 and TRAF5 are closely related members of the TRAF family of proteins. They are important signal transducers for a wide range of TNF receptor superfamily members, including TNFR1, TNFR2, CD40 and other lymphocyte costimulatory receptors, RANK/TRANCE-R, EDAR, LTbetaR, LMP-1 and IRE1. TRAF2 and TRAF5 therefore regulate diverse physiological roles, ranging from T and B cell signaling and inflammatory responses to organogenesis and cell survival. The major pathways mediated by TRAF2 and TRAF5 are the classical and alternative pathways of NF-kappaB activation, and MAPK and JNK activation. TRAF2 is heavily regulated by ubiquitin signals, and many of the signaling functions of TRAF2 are mediated through its RING domain and likely its own role as an E3 ubiquitin ligase.

2/7/13 (Item 2 from file: 72)
DIALOG(R)File 72:EMBASE

0080455577 EMBASE No: 2005099733

TNF receptor (TNFR)-associated factor (TRAF) 3 serves as an inhibitor of TRAF2/5-mediated activation of the noncanonical NF-kappaB pathway by TRAF-binding TNFRs

Hauer J.; Puschner S.; Ramakrishnan P.; Simon U.; Bongers M.; Federle C.; Engelmann H.
Institut fur Immunologie, Universitat Munchen, Goethestrasse 31, 80366 Munich, Germany
AUTHOR EMAIL: hengelmann@lmu.de:
CORRESP. AUTHOR/AFFIL: Institut fur Immunologie, Universitat Munchen, Goethestrasse 31, 80366 Munich, Germany
CORRESP. AUTHOR EMAIL: hengelmann@lmu.de

Proceedings of the National Academy of Sciences of the United States of America (Proc. Natl. Acad. Sci. U. S. A.) (United States) February 22, 2005, 102/8 (2874-2879)
CODEN: PNASA ISSN: 0027-8424
DOI: 10.1073/pnas.0500187102
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 46

TNF family members and their receptors contribute to increased gene expression for inflammatory processes and intracellular cascades leading to programmed cell death, both via activation of NF-kappaB. TNF receptor (TNFR)-associated factors (TRAFs) are cytoplasmic adaptor proteins binding to various receptors of the TNFR family. In an attempt to delineate the role of individual TRAFs, we compared NF-kappaB activation by CD40 SUB wt and CD40 mutants with different TRAF recruitment patterns. Recognized only recently, NF-kappaB signaling occurs at least via two different pathways. Each pathway results in nuclear translocation of two different Rel-dimers, the canonical p50/RelA and the noncanonical p52/RelB. Here, we show that via TRAF6, CD40 mediates only the activation of the canonical NF-kappaB pathway. Via TRAF2/5, CD40 activates both the canonical and the noncanonical NF-kappaB pathways. We observed that TRAF3 specifically blocked the NF-kappaB activation via TRAF2/5. This inhibitory effect of TRAF3 depends on the presence of an intact zinc finger domain.

Paradoxically, suppression of TRAF2/5-mediated NF-kappaB activation by TRAF3 resulted in enhanced transcriptional activity of TRAFS-mediated canonical NF-kappaB emanating from CD40. We also observed that 12 TNFR family members (p75TNFR, LTbetaR, RANK, HVEM, CD40, CD30, CD27, 4-1BB, GITR, BCMA, OX40, and TACl) are each capable of activating the alternative NF-kappaB pathway and conclude that TRAF3 serves as a negative
regulator of this pathway for all tested receptors.

2/7/14 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

28253583 PMID: 16572839
[Cloning, soluble expression and characterization of human sBCMA]
Guan Zheng-Bing; Cao Peng; Ye Ji-Lin; Zhang Shuang-Quan
Jiangsu Province Key Laboratoryfor Molecular and Medical Biotechnology,
Life Sciences College, Nanjing Normal University, Nanjing 210097, China.
Sheng wu gong cheng xue bao = Chinese journal of biotechnology (China)
Jan 2006, 22 (1) p46-51, ISSN 1000-3061--Print Journal Code: 9426463
Publishing Model Print
Document type: English Abstract; Journal Article; Research Support, Non-U.S. Gov't
Languages: CHINESE
Main Citation Owner: NLM
Record type: In Process
BCMA is one of the transmembrane receptors belonging to BAFF and APRIL. In order to identify the feasibility of sBCMA as decoy receptor
and obtain active sBCMA for its structural and functional research, full
length of hBCMA was amplified with total RNA from Raji cell line by RT-PCR,
and the cDNA encoding the extracelluar soluble domain of hBCMA was inserted into pET43.1a(+) vector. The recombinant vector pET43.1a(+)-sBCMA
was transformed into E. coli Origami B(DE3) pLyS which is helpful for
disulfide bond construction of expression proteins. After IPTG induction,
the recombinant protein was expressed as soluble fusion protein,
sBCMA-NusA-His6, and identified by western blotting. Then the target
protein was purified by Ni(+)-chelating Sepharose Fast Flow. The binding

activity between recombinant sBCMA and BAFF was detected by ELISA. Also,
Recombinant sBCMA inhibited proliferation of mouse B cell stimulating by
rhsBAFF. It was proved that recombinant sBCMA has good bioactivity and the
method to express those proteins rich in disulfide bond is feasible and
effectual.
Record Date Created: 20060331

2/7/15 (Item 2 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

16421452 PMID: 15542592
Structures of APRIL-receptor complexes: like BCMA, TACI employs only a single cysteine-rich domain for high affinity ligand binding.
Hymowitz Sarah G; Patel Darshana R; Wallweber Heidi J A; Runyon Steven;
Yan Minhong; Yin Jianping; Shriver Stephanie K; Gordon Nathaniel C; Pan
Borlan; Skelton Nicholas J; Kelley Robert F; Starovasnik Melissa A
Department of Protein Engineering, Molecular Oncology, Medicinal
Chemistry, and Immunology, Genentech, Inc., South San Francisco, California
94080, USA.
Journal of biological chemistry (United States) Feb 25 2005, 280 (8)
p7218-27, ISSN 0021-9258--Print Journal Code: 2985121R
Publishing Model Print-Electronic
Document type: Journal Article; Research Support, U.S. Gov't, Non-P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
TACI is a member of the tumor necrosis factor receptor superfamily and
serves as a key regulator of B cell function. TACI binds two ligands, APRIL
and BAFF, with high affinity and contains two cysteine-rich domains (CRDs)
in its extracellular region; in contrast, BCMA and BR3, the other known high affinity receptors for APRIL and BAFF, respectively, contain
only a single or partial CRD. However, another form of TACI exists wherein
the N-terminal CRD is removed by alternative splicing. We find that this
shorter form is capable of ligand-induced cell signaling and that the

second CRD alone (TACI d2) contains full affinity for both ligands.
Furthermore, we report the solution structure and alanine-scanning
mutagenesis of TACI d2 along with co-crystal structures of APRIL.TACI d2
and APRIL.BCMA complexes that together reveal the mechanism by which
TACI engages high affinity ligand binding through a single CRD, and we
highlight sources of ligand-receptor specificity within the APRIL/BAFF
system.
Record Date Created: 20050221
Record Date Completed: 20050408
Date of Electronic Publication: 20041112

2/7/16 (Item 3 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

15230933 PMID: 12594954
Loss of TACI causes fatal lymphoproliferation and autoimmunity,
establishing TACI as an inhibitory BLyS receptor.
Seshasayee Dhaya; Valdez Patricia; Yan Minhong; Dixit Vishva M; Tumas
Daniel; Grewal Iqbal S
Department of Immunology, Genentech, Inc, 1 DNA Way, South San Francisco,
CA 94080, USA.
Immunity (United States) Feb 2003, 18 (2) p279-88, ISSN 1074-7613--
Print Journal Code: 9432918
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BLys , a key cytokine that sustains B cell maturation and tolerance,
binds three receptors: BR3, BCMA, and TACI. Results from knockout mice
implicate a major functional role for BR3 and a redundant one for
BCMA in B cell function. TACI's role is controversial based on defects
in TI antibody responses accompanied by B cell hyperplasia in
knockout mice. We have presently characterized a precise role for TACI in
vivo. TACI(-/-) mice develop fatal autoimmune glomerulonephritis,
proteinurea, and elevated levels of circulating autoantibodies.
Treatment

of B cells with TACI agonistic antibodies inhibits proliferation in vitro
and activation of a chimeric receptor containing the TACI intracellular
domain induces apoptosis. These results demonstrate the critical requirement for TACI in regulating B cell homeostasis.
Record Date Created: 20030221
Record Date Completed: 20030325

2/7/17 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133103739 CA: 133(8)103739s PATENT
Soluble receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in modulating the
immune response and treating autoimmune disorders
INVENTOR(AUTHOR): Gross, Jane A.; Xu, Wenfeng; Madden, Karen; Yee, David
P.
LOCATION: USA
ASSIGNEE: Zymogenetics, Inc.
PATENT: PCT International ; WO 200040716 A2 DATE: 20000713
APPLICATION: WO 2000US396 (20000107) *US 226533 (19990107)
PAGES: 175 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C12N-015/11A; C07K-014/705B; A61K-038/17B; A61K-039/395B
DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH;
CN; CR; CU; CZ; DE; DK; DM; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL;
IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN;
MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA;
UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE;
CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF;
CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA215010 Immunochemistry
IDENTIFIERS: TACI isoform BR43x2 immune modulation autoimmune disorder,
sequence BR43x2 protein cDNA human mouse, BCMA protein immune
modulation autoimmune disorder
DESCRIPTORS:
Proteins,specific or class...
BCMA (B cell membrane antigen); sol. receptor BR43x2 isoform of

transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating autoi
Bronchi...
bronchitis, treatment of; sol. receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune
Proteins,specific or class...
BR43x2; sol. receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in modulating
the immune response and treating autoimmune disorders
Proteins,specific or class...
CAML (calcium-modulator and cyclophilin ligand); sol. receptor BR43x2
isoform of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response a
Intestine...
colon, expression specificity in; sol. receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating aut
Intestine,disease...
Crohn's, treatment of; sol. receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune dis
Joint,anatomical...
disease, treatment of; sol. receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune dis
Appendix... B cell(lymphocyte)... Bone marrow... Lung... Lymph node...
Lymphoma... Salivary gland... Spleen... Stomach... Testis...
Trachea(anatomical)...
expression specificity in; sol. receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating autoimmune
Immunoglobulins...
fusion products; sol. receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune disorders

Transplant and Transplantation...
    graft-vs.-host reaction, treatment of; sol. receptor BR43x2 isoform of
    transmembrane activator and CAML-interactor TACI and related proteins
    and their use in modulating the immune response and treatin
Antibodies...
    humanized; sol. receptor BR43x2 isoform of transmembrane activator and
    CAML-interactor TACI and related proteins and their use in modulating
    the immune response and treating autoimmune disorders
Diabetes mellitus...
    insulin-dependent, treatment of; sol. receptor BR43x2 isoform of
    transmembrane activator and CAML-interactor TACI and related proteins
    and their use in modulating the immune response and treating auto
Tumor necrosis factors...
    ligand neutrokine α; sol. receptor BR43x2 isoform of
    transmembrane activator and CAML-interactor TACI and related proteins
    and their use in modulating the immune response and treating autoimmune
Antibodies...
    monoclonal; sol. receptor BR43x2 isoform of transmembrane activator and
    CAML-interactor TACI and related proteins and their use in modulating
    the immune response and treating autoimmune disorders
Nerve,disease...
    neuropathy, Ig light chain, treatment of; sol. receptor BR43x2 isoform
    of transmembrane activator and CAML-interactor TACI and related
    proteins and their use in modulating the immune response and trea
Cytokines...
    neutrokine α; sol. receptor BR43x2 isoform of transmembrane
    activator and CAML-interactor TACI and related proteins and their use
    in modulating the immune response and treating autoimmune disord
Lymphoma...
    non-Hodgkin's, expression specificity in; sol. receptor BR43x2 isoform
    of transmembrane activator and CAML-interactor TACI and related
    proteins and their use in modulating the immune response and trea
Salivary gland...
    parotid, neoplasm, expression specificity in; sol. receptor BR43x2
    isoform of transmembrane activator and CAML-interactor TACI and related
    proteins and their use in modulating the immune response and
Animal cell line...
    Raji, expression specificity in; sol. receptor BR43x2 isoform of
    transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating auto
Shock(circulatory collapse)...
septic, treatment of; sol. receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune diso
Intestine...
small, expression specificity in; sol. receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating aut
Antibodies... cDNA sequences... Drugs... Gene therapy...
Immunosuppression
... Mammal(Mammalia)... Molecular cloning... Mouse... Primate...
Protein
sequences...
sol. receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in
modulating
the immune response and treating autoimmune disorders
Lupus erythematosus...
systemic, treatment of; sol. receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune di
Proteins,specific or class...
TACI (transmembrane activator and CAML-interactor); sol. receptor
BR43x2 isoform of transmembrane activator and CAML-interactor
TACI and
related proteins and their use in modulating the immune respons
Amyloidosis... Anemia(disease)... Asthma... Autoimmune disease...
Emphysema
... Inflammation... Kidney,disease... Kidney,neoplasm... Multiple
myeloma
... Multiple sclerosis... Myasthenia gravis... Rheumatoid arthritis...
Swelling,biological... Transplant rejection...
treatment of; sol. receptor BR43x2 isoform of transmembrane
activator
and CAML-interactor TACI and related proteins and their use in
modulating the immune response and treating autoimmune disorders
Fusion proteins(chimeric proteins)...
with Ig heavy chain; sol. receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune disor
CAS REGISTRY NUMBERS:
217638-65-8 282738-49-2 amino acid sequence; sol. receptor BR43x2
isoform
of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and
treating

autoimmune disorders
283157-99-3 cysteine-rich pseudo repeat domain; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
156253-82-6 198123-04-5 282738-43-6 282738-45-8 282738-47-0 282738-48-1 nucleotide sequence; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
148997-64-2D 198029-64-0D 282738-44-7D 282738-46-9D subfragments are claimed, amino acid sequence; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
225456-59-7 271755-95-4 274950-83-3 274950-84-4 282738-77-6 282738-78-7 282738-79-8 282738-80-1 282738-81-2 282738-82-3 282738-83-4 282738-84-5 282738-85-6 282738-86-7 282738-87-8 282738-88-9 282738-89-0 282738-90-3 282738-91-4 282738-92-5 282738-93-6 282738-94-7 282738-95-8 282738-96-9 282738-97-0 282738-98-1 282738-99-2 282739-00-8 282739-02-0 282739-03-1 282739-04-2 282739-05-3 282739-06-4 282739-07-5 282739-08-6 282739-09-7 282739-10-0 282739-11-1 282739-12-2 282739-13-3 unclaimed nucleotide sequence; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
282738-76-5 282739-01-9 unclaimed protein sequence; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
98849-88-8 256922-04-0 282729-02-6 282729-03-7 unclaimed sequence; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
? ds

Set Items Description

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S1          62   TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION AN-
              TIGEN)
S2          17   RD S1  (unique items)
? ds

Set     Items   Description
S1          62   TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION AN-
              TIGEN)
S2          17   RD S1  (unique items)
? s BCMA or (b(w) cell(w) maturation (w) antigen)
Processing
Processed  10 of  29 files ...
Processing
Processing
Processed  20 of  29 files ...
Processing
Completed processing all files
            1556  BCMA
         9559397  B
        22153748  CELL
          779077  MATURATION
         3353699  ANTIGEN
             643  B(W)CELL(W)MATURATION(W)ANTIGEN
      S3    1744  BCMA OR (B(W) CELL(W) MATURATION (W) ANTIGEN)
? s soluble and s3
         1456508  SOLUBLE
            1744  S3
      S4     280  SOLUBLE AND S3
? rd s4

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
      S5      73  RD S4  (unique items)
? s s5 not PY>2001
Processing
Processed  10 of  29 files ...
Processing
Processed  20 of  29 files ...
Completed processing all files
              73  S5
        54517690  PY>2001
      S6      13  S5 NOT PY>2001
? t s6/7/all
>>>Format 7 is not valid in file 143

 6/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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16667984 BIOSIS NO.: 200200261495
Therapeutic potential of antagonizing BLyS for chronic lymphocytic leukemia
AUTHOR: Zhou Tong (Reprint); Liu Weimin (Reprint); Zhao Limin (Reprint);
Carter Robert H (Reprint); Kimberly Robert P (Reprint); Emanuel Peter D
AUTHOR ADDRESS: Division of Clinical Immunology and Rheumatology, University of Alabama at Birmingham, Birmingham, AL, USA**USA
JOURNAL: Blood 98 (11 Part 1): p808a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The pathogenesis of the accumulation of malignant B cells with a mature phenotype in CLL is poorly understood, and curative therapies for CLL do not yet exist. B Lymphocyte Stimulator (BLyS) is a newly identified B cell survival factor of the TNF superfamily, which plays a crucial role in B cell development and autoimmune disease. At least three receptors for BLyS have been identified: TACI, BCMA and BAFF-R. To determine the role of BLyS and its receptors in CLL, we examined expression of two receptors: TACI and BCMA using newly generated monoclonal antibodies. Expression of cell surface BLyS with TACI-Fc fusion protein; and expression of soluble BLyS by ELISA was examined in primary peripheral blood cells from 11 patients with CLL.
Similar to normal B cells, the B cells from all CLL patients expressed high levels of BLyS binding receptors. However, while normal B cells did not express significant levels of either TACI or BCMA, the CLL B cells from >50% (6/11) and 80% (9/11) patients expressed increased levels of TACI and BCMA, respectively, indicating that expression of TACI and BCMA is upregulated in CLL. Normal B cells did not express cell surface BLyS as detected by TACI-Fc fusion protein. In contrast, the CLL B cells from >80% (9/11) had higher levels of cell surface BLyS, which was confirmed by RT-PCR and Western blot analysis using BLyS specific primers and antibody. These results suggest that a positive autocrine

loop through cell surface BLyS and its receptors might play a crucial role in the survival and accumulation of CLL B cells. Surprisingly, serum levels of BLyS were almost undetectable in all patients with CLL, suggesting that the circulating BLyS might be over-consumed by the CLL B cells. Compared to normal B cells, the CLL B cells had a poorer response to stimulation with exogenous BLyS in vitro, suggesting that endogenous BLyS is sufficient to sustain the survival of CLL B cells. However, in vitro treatment with TACI-Fc and a BLyS neutralizing monoclonal antibody (15c10) resulted in decreased survival of the CLL B cells in a time-dependent fashion. To further determine the therapeutic potential of blocking BLyS, NK-depleted NOD/SCID mice were reconstituted with the CLL B cells and treated with three doses of 100mug TACI-Fc. Seven days after transfer, the CLL B cells were recovered from the spleens of the recipient mice. The CLL B cells from all 11 patients exhibited the ability to repopulate in the spleens of untreated SCID mice as determined by CD19+ cell number. Treatment with isotype control IgG1 did not alter the cell number recovered from the spleen. However, the number of CLL B cells isolated from majority of patients (8/11) was significantly reduced (50%-85% reduction) in the spleens of recipient mice treated with TACI-Fc. Taken together, our results indicate that BLyS and its receptors, TACI and BCMA, play a crucial role in the survival and accumulation of malignant B cells in CLL patients. Thus, blockade of BLyS stimulation with TACI-Fc or BLyS neutralizing antibody may prove to be an effective treatment for CLL.

6/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16459180 BIOSIS NO.: 200200052691
Maturation of marginal zone and follicular B cells requires B cell activating factor of the tumor necrosis factor family and is independent of B cell maturation antigen
AUTHOR: Schneider Pascal; Takatsuka Hisakazu; Wilson Anne; MacKay Fabienne;

Tardivel Aubry; Lens Susanne; Cachero Teresa G; Finke Daniela; Beermann
Friedrich; Tschopp Jurg (Reprint)
AUTHOR ADDRESS: Institute of Biochemistry, University of Lausanne, Ch. des
Boveresses 155, CH-1066, Epalinges, Switzerland**Switzerland
JOURNAL: Journal of Experimental Medicine 194 (11): p1691-1697 December 3,
2001 2001
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: B cells undergo a complex series of maturation and selection steps in the bone marrow and spleen during differentiation into mature immune effector cells. The tumor necrosis factor (TNF) family member B cell activating factor of the TNF family (BAFF) (BLyS/TALL-1) plays an important role in B cell homeostasis. BAFF and its close homologue a proliferation-inducing ligand (APRIL) have both been shown to interact with at least two receptors, B cell maturation antigen (BCMA) and transmembrane activator and cyclophilin ligand interactor (TACI), however their relative contribution in transducing BAFF signals in vivo remains unclear. To functionally inactivate both BAFF and APRIL, mice transgenic for a soluble form of TACI were generated. They display a developmental block of B cell maturation in the periphery, leading to a severe depletion of marginal zone and follicular B2 B cells, but not of peritoneal B1 B cells. In contrast, mice transgenic for a soluble form of BCMA, which binds APRIL, have no detectable B cell phenotype. This demonstrates a crucial role for BAFF in B cell maturation and strongly suggests that it signals via a BCMA-independent pathway and in an APRIL-dispensable way.

16143164 BIOSIS NO.: 200100315003
Blocking of BAFF signaling pathway by BCMA-Fc attenuates autoimmune manifestations in BAFF transgenic mice and reveals its important role in maintaining peripheral B cell homeostasis

AUTHOR: Woodcock Stephen A (Reprint); Liu Zhong-Ying (Reprint); Cachero
  Teresa G (Reprint); Xian Fang (Reprint); Thill Greg (Reprint); Ambrose
  Christine (Reprint); Thompson Jeffery S (Reprint); Spinello Nicole
  (Reprint); Mackay Fabienne; Browning Jeffrey L (Reprint); Kalled Susan L
  (Reprint); Wang LiChun (Reprint)
AUTHOR ADDRESS: Immunology and Inflammation, Biogen Inc., Cambridge, MA,
  USA**USA
JOURNAL: Blood 96 (11 Part 1): p616a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English
ABSTRACT: We have shown previously that the B cell
  maturation antigen (BCMA) is a member of the TNF
  receptor family and binds to B cell activating factor (BAFF) expressed by
  cells of dendritic and myeloid lineages. Treatment of normal mice with a
  soluble BCMA-Fc decoy, comprised of the extracellular domain
  of human BCMA fused to the human IgG1 hinge, CH2 and CH3 domains,
  resulted in the progressive loss of peripheral B cell subpopulations
  including transitional(T)1, T2, mature and marginal zone cells. Bone
  marrow hematopoiesis, however, was not affected. The reduction of
  peripheral B cells by BCMA-Fc treatment is not Fc-dependent.
  Additionally, treatment with BCMA-Fc in Baff transgenic mice
  resulted in a reduction of elevated numbers of B cells, splenomegaly, and
  proteinuria, all hallmark disease phenotypes of BAFF transgenic mice.
  Taken together, the data provide in vivo evidence for the utility of
  BCMA-Fc for treatment of diseases characterized by B cell
  disfunction.

6/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16086684 BIOSIS NO.: 200100258523
TALL-1 is a target for B cell mediated autoimmune diseases in mice and
  human
AUTHOR: Miner Kent T; Eastman Susan; Xia Xing Z; McCabe Susan; Hawkins
  Nissa; Boone Tom; Delaney John; Lee Francis; Hsu Hailing; Khare Sanjay D

JOURNAL: FASEB Journal 15 (5): pA1212 March 8, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies
for Experimental Biology on Experimental Biology 2001 Orlando, Florida,
USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Members belonging to TNF superfamily are important in various
aspects related to cell proliferation and death during immune regulation.
A recently discovered member of the TNF family, TALL-1
(Blys/BAFF/THANK/zTNF4) is involved in B cell proliferation and antibody
production. Lupus prone NZBxNZWF1 mice at 2 months of age developed
autoantibodies to ds-DNA and histone proteins when injected with
recombinant soluble TALL-1 protein. Transgenic mice over expressing
TALL-1 in non-lupus prone background showed increased antibody production
and developed lupus like disease. TACI and BCMA have recently been
identified as receptors for TALL-1. We examined a therapeutic effect of
soluble receptors in lupus prone animals. Development of
proteinurea (>300mg/dl) was delayed in both lupus prone MRL/lpr-lpr and
NZBWF1 mice when treated with a recombinant soluble receptor fusion
protein, TACI-Fc. TACI treatment also prolonged the survival time. We
further examined serum levels of TALL-1 in several human autoimmune
diseases. Patients with systemic lupus erythematosus, Myasthenia gravis
and Wegener's granulomatosus showed significantly elevated levels of
TALL-1 in serum when compared with healthy controls. Taken together, our
data strongly suggest that TALL-1 is an important target in B cell
mediated autoimmune diseases.

6/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15922018 BIOSIS NO.: 200100093857
APRIL and TALL-I and receptors BCMA and TACI: System for regulating
humoral immunity
AUTHOR: Yu Gang; Boone Tom; Delaney John; Hawkins Nessa; Kelley Michael;

Ramakrishnan Meena; McCabe Susan; Qiu Wan-rong; Kornuc Masayo; Xia Xing-Zhong; Guo Jane; Stolina Marina; Boyle William J; Sarosi Ildiko; Hsu Hailing; Senaldi Giorgio; Theill Lars E (Reprint)
AUTHOR ADDRESS: Department of Inflammation, Amgen Inc., One Amgen Center Drive, Thousand Oaks, CA, 91320-1799, USA**USA
JOURNAL: Nature Immunology 1 (3): p252-256 September, 2000 2000
MEDIUM: print
ISSN: 1529-2908
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We report that the tumor neurosis factor homolog APRIL (a proliferation-inducing ligand) stimulates in vitro proliferation of primary B and T cells and increases spleen weight due to accumulation of B cells in vivo. APRIL functions via binding to BCMA (B cell maturation antigen) and TACI (transmembrane activator and CAML-interactor) and competes with TALL-I (also called BLyS or BAFF) for receptor binding. Soluble BCMA and TACI specifically prevent binding of APRIL and block APRIL-stimulated proliferation of primary B cells. BCMA-Fc also inhibits production of antibodies against keyhole limpet hemocyanin and Pneumovax in mice, indicating that APRIL and/or TALL-I signaling via BCMA and/or TACI are required for generation of humoral immunity. Thus, APRIL-TALL-I and BCMA-TACI form a two ligands-two receptors pathway involved in stimulation of B and T cell function.

6/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15897239 BIOSIS NO.: 200100069078
A soluble form of B cell maturation antigen, a receptor for the tumor necrosis factor family member APRIL, inhibits tumor cell growth
AUTHOR: Rennert Paul; Schneider Pascal; Cachero Teresa G; Thompson Jeffrey; Trabach Luciana; Hertig Sylvie; Holler Nils; Qian Fang; Mullen Colleen; Strauch Kathy; Browning Jeffrey L; Ambrose Christine; Tschopp Jurg (Reprint)
AUTHOR ADDRESS: Institute of Biochemistry, University of Lausanne, Ch. des Boveresses 155, CH-1066, Epalinges, Switzerland**Switzerland

JOURNAL: Journal of Experimental Medicine 192 (11): p1677-1683 December 4, 2000 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A proliferation-inducing ligand (APRIL) is a ligand of the tumor necrosis factor (TNF) family that stimulates tumor cell growth in vitro and in vivo. Expression of APRIL is highly upregulated in many tumors including colon and prostate carcinomas. Here we identify B cell maturation antigen (BCMA) and transmembrane activator and calcium modulator and cyclophilin ligand (CAML) interactor (TACI), two predicted members of the TNF receptor family, as receptors for APRIL. APRIL binds BCMA with higher affinity than TACI. A soluble form of BCMA, which inhibits the proliferative activity of APRIL in vitro, decreases tumor cell proliferation in nude mice. Growth of HT29 colon carcinoma cells is blocked when mice are treated once per week with the soluble receptor. These results suggest an important role for APRIL in tumorigenesis and point towards a novel anticancer strategy.

6/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15720104 BIOSIS NO.: 200000438417
B cell maturation protein is a receptor for the tumor necrosis factor family member TALL-1
AUTHOR: Shu Hong-Bing (Reprint); Johnson Holly
AUTHOR ADDRESS: Department of Immunology, National Jewish Medical and Research Center and University of Colorado School of Medicine, 1400 Jackson Street, K516c, Denver, CO, 80206, USA**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (16): p9156-9161 August 1, 2000 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: TALL-1 is a recently identified member of the tumor necrosis

factor (TNF) family that costimulates B lymphocyte proliferation. Here we show that B cell maturation protein (BCMA), a member of the TNF receptor family that is expressed only by B lymphocytes, specifically binds to TALL-1. A soluble receptor containing the extracellular domain of BCMA blocks the binding of TALL-1 to its receptor on the plasma membrane and inhibits TALL-1-triggered B lymphocyte costimulation. Overexpression of BCMA activates NF-kappaB, and this activation is potentiated by TALL-1. Moreover, BCMA-mediated NF-kappaB activation is inhibited by dominant negative mutants of TNF receptor-associated factor 5 (TRAF5), TRAF6, NF-kappaB-inducing kinase (NIK), and IkappaB kinase (IKK). These data indicate that BCMA is a receptor for TALL-1 and BCMA activates NF-kappaB through a TRAF5-, TRAF6-, NIK-, and IKK-dependent pathway. The identification of BCMA as a NF-kappaB-activating receptor for TALL-1 suggests molecular targets for drug development against certain immunodeficient or autoimmune diseases.

6/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15658954 BIOSIS NO.: 200000377267
BAFF binds to the tumor necrosis factor receptor-like molecule B cell maturation antigen and is important for maintaining the peripheral B cell population
AUTHOR: Thompson Jeffrey S; Schneider Pascal; Kalled Susan L; Wang LiChun; Lefevre Eric A; Cachero Teresa G; MacKay Fabienne; Bixler Sarah A; Zafari Mohammad; Liu Zhong-Ying; Woodcock Stephen A; Qian Fang; Batten Marcel; Madry Christine; Richard Yolande; Benjamin Christopher D; Browning Jeffrey L; Tsapis Andreas; Tschopp Jurg; Ambrose Christine (Reprint)
AUTHOR ADDRESS: Biogen, Inc., 12 Cambridge Center, Cambridge, MA, 02142, USA**USA
JOURNAL: Journal of Experimental Medicine 192 (1): p129-135 July 3, 2000
2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The tumor necrosis factor (TNF) family member B cell activating

factor (BAFF) binds B cells and enhances B cell receptor-triggered proliferation. We find that B cell maturation antigen (BCMA), a predicted member of the TNF receptor family expressed primarily in mature B cells, is a receptor for BAFF. Although BCMA was previously localized to the Golgi apparatus, BCMA was found to be expressed on the surface of transfected cells and tonsillar B cells. A soluble form of BCMA, which inhibited the binding of BAFF to a B cell line, induced a dramatic decrease in the number of peripheral B cells when administered in vivo. Moreover, culturing splenic cells in the presence of BAFF increased survival of a percentage of the B cells. These results are consistent with a role for BAFF in maintaining homeostasis of the B cell population.

6/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15587774 BIOSIS NO.: 200000306087
TACI and BCMA are receptors for a TNF homologue implicated in B-cell autoimmune disease
AUTHOR: Gross Jane A (Reprint); Johnston Janet; Mudri Sherri; Enselman Rachel; Dillon Stacey R; Madden Karen; Xu Wenfeng; Parrish-Novak Julia; Foster Don; Lofton-Day Cathy; Moore Margaret; Littau Alisa; Grossman Angelika; Haugen Harald; Foley Kevin; Blumberg Hal; Harrison Kim; Kindsvogel Wayne; Clegg Christopher H
AUTHOR ADDRESS: Department of Immunology, ZymoGenetics, 1201 Eastlake Avenue East, Seattle, WA, 98102, USA**USA
JOURNAL: Nature (London) 404 (6781): p995-999 April 27, 2000 2000
MEDIUM: print
ISSN: 0028-0836
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: B cells are important in the development of autoimmune disorders by mechanisms involving disregulated polyclonal B-cell activation, production of pathogenic antibodies, and co-stimulation of autoreactive T cells. zTNF4 (BLyS, BAFF, TALL-1, THANK) is a member of the tumour necrosis factor (TNF) ligand family that is a potent co-activator of B cells in vitro and in vivo. Here we identify two receptors for zTNF4 and demonstrate a relationship between zTNF4 and autoimmune disease. Transgenic animals overexpressing zTNF4 in lymphoid cells develop

symptoms characteristic of systemic lupus erythaematosus (SLE) and expand a rare population of splenic B-1a lymphocytes. In addition, circulating zTNF4 is more abundant in NZBWFI and MRL-lpr/lpr mice during the onset and progression of SLE. We have identified two TNF receptor family members. TACI and BCMA, that bind zTNF4. Treatment of NZBWF1 mice with soluble TACI-Ig fusion protein inhibits the development of proteinuria and prolongs survival of the animals. These findings demonstrate the involvement of zTNF4 and its receptors in the development of SLE and identify TACI-Ig as a promising treatment of autoimmune disease in humans.

6/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

06131156 BIOSIS NO.: 198121015119
PRESENT ASPECTS OF LYMPHOCYTE ONTOGENESIS AND DIFFERENTIATION
AUTHOR: ASTALDI G (Reprint); TOPUZ U O; NERI A; IACOPINO P
AUTHOR ADDRESS: IST SIEROTERAPICO MILANESE S BELFANTI, VIA DARWIN, 20/22- 20143 MILANO**ITALY
JOURNAL: Bollettino dell'Istituto Sieroterapico Milanese 59 (4): p255-292
1980
ISSN: 0021-2547
DOCUMENT TYPE: Article
RECORD TYPE: Citation
LANGUAGE: ITALIAN

6/7/11 (Item 1 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002291577 IP ACCESSION NO: 5313257
Polymorphism and chromosomal mapping of the mouse gene for B-cell activating factor belonging to the tumor necrosis factor family (Baff) and association with the autoimmune phenotype

Jiang, Y; Ohtsuji, M; Abe, M; Li, N; Xiu, Y; Wen, XF; Shirai, T; Hirose, S
Department of Pathology, Juntendo University School of Medicine, 2-1-1 Hongo, Bunkyo-ku, Tokyo 113-8421, Japan

Immunogenetics, v 53, n 9, p 810-813, December 2001
PUBLICATION DATE: 2001

PUBLISHER: Springer-Verlag,
[URL:http://link.springer.de/link/service/journals/00251/bibs/1053009/10530810.htm]

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
ISSN: 0093-7711
DOI: 10.1007/s00251-001-0396-6
FILE SEGMENT: Genetics Abstracts; Immunology Abstracts

ABSTRACT:
B-cell activating factor (BAFF), also known as BlyS, THANK, and zTNF4, is a new member of the tumor necrosis factor (TNF) family that is constitutively produced by macrophages, dendritic cells, and activated T cells. BAFF is cleaved by furin protease, and the product is shed into the bloodstream. The cleaved BAFF product binds its receptor, TACI and BCMA specifically expressed on B cells and activates B-lineage cells. Elevated BAFF, as a result of injection or transgene expression, causes B-cell lymphadenopathy, CD5+ B1a cell expansion, plasmacytosis, or a systemic lupus erythematosus (SLE)-like autoimmune disease. These phenotypes appear to be due to up-regulation of antiapoptotic Bcl-2 expression in B cells, since B cells in BAFF-transgenic mice express high levels of Bcl-2. Relevant are findings that SLE-like disease occurs in mice expressing a bcl-2 transgene and in mice lacking the proapoptotic Bcl-2 family member Bim. SLE is a multifactorial autoimmune disease and much of the related pathology can be attributed to immune complexes formed by pathogenic high-affinity autoantibodies, including those against nuclear components. Genes that predispose to SLE are related to processes of emergence, activation, clonal expansion, differentiation, and maturation of autoreactive B cells. Such being the case, abnormalities in Baff may lead to the pathogenesis of SLE. (NZB x NZW)F1 mice are genetically susceptible to SLE and during aging spontaneously develop the disease. Gross and co-workers reported a strong correlation between disease progression and the amount of circulating serum BAFF (zTNF4) in (NZB x NZW)F1 mice. Because treatment of (NZB x NZW)F1 mice with soluble TACI-Ig fusion protein inhibited the development of immune complex-type glomerulonephritis (lupus

nephritis), and prolonged survival of the animals, BAFF may be a primary
mediator of B cell-associated autoimmune disease in these mice. If this
notion is tenable, the Baff allele of either NZB or NZW may be polymorphic.

6/7/12 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09831500 Genuine Article#: 453UW Number of References: 20
Title: The role of TALL-1 and APRIL in immune regulation
Author(s): Khare SD (REPRINT) ; Hsu HL
Corporate Source: Amgen Inc,Dept Pathol Pharmacol,1 Amgen Ctr Dr/Thousand
Oaks//CA/91320 (REPRINT); Amgen Inc,Dept Pathol Pharmacol,Thousand
Oaks//CA/91320; Amgen Inc,Dept Inflammat,Thousand Oaks//CA/91320
Journal: TRENDS IN IMMUNOLOGY, 2001, V22, N2 (FEB), P61-63
ISSN: 1471-4906 Publication date: 20010200
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON,
OXFORD OX5 1GB, OXON, ENGLAND
Language: English Document Type: EDITORIAL MATERIAL
Abstract: Members of the tumor necrosis factor (TNF) superfamily play important roles in cell proliferation and death during immune regulation. Most members are synthesized as type II transmembrane proteins; the carboxy terminal extracellular domain can be cleaved from the cell membrane to form soluble active cytokines that bind to appropriate members of the TNF receptor family Here, we describe the biological significance of recently discovered members of the TNF superfamily (TALL-1 and APRIL) and their receptors (TACI and BCMA) in the pathophysiology of human diseases.

6/7/13 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

133103739 CA: 133(8)103739s PATENT
Soluble receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in modulating the
immune response and treating autoimmune disorders
INVENTOR(AUTHOR): Gross, Jane A.; Xu, Wenfeng; Madden, Karen; Yee, David
P.
LOCATION: USA
ASSIGNEE: Zymogenetics, Inc.
PATENT: PCT International ; WO 200040716 A2 DATE: 20000713

APPLICATION: WO 2000US396 (20000107) *US 226533 (19990107)
PAGES: 175 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C12N-015/11A; C07K-014/705B; A61K-038/17B; A61K-039/395B
DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH;
CN; CR; CU; CZ; DE; DK; DM; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL;
IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN;
MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA;
UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE;
CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF;
CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA215010 Immunochemistry
IDENTIFIERS: TACI isoform BR43x2 immune modulation autoimmune disorder,
sequence BR43x2 protein cDNA human mouse, BCMA protein immune
modulation autoimmune disorder
DESCRIPTORS:
Proteins,specific or class...
BCMA (B cell membrane antigen); soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating autoi
Bronchi...
bronchitis, treatment of; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune
Proteins,specific or class...
BR43x2; soluble receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in modulating
the immune response and treating autoimmune disorders
Proteins,specific or class...
CAML (calcium-modulator and cyclophilin ligand); soluble receptor BR43x2
isoform of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response a
Intestine...
colon, expression specificity in; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating aut
Intestine,disease...
Crohn's, treatment of; soluble receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune dis
Joint,anatomical...
disease, treatment of; soluble receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune dis
Appendix... B cell(lymphocyte)... Bone marrow... Lung... Lymph node...
Lymphoma... Salivary gland... Spleen... Stomach... Testis...
Trachea(anatomical)...
expression specificity in; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating
autoimmune
Immunoglobulins...
fusion products; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune
disorders
Transplant and Transplantation...
graft-vs.-host reaction, treatment of; soluble receptor BR43x2
isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treatin
Antibodies...
humanized; soluble receptor BR43x2 isoform of transmembrane
activator and
CAML-interactor TACI and related proteins and their use in
modulating
the immune response and treating autoimmune disorders
Diabetes mellitus...
insulin-dependent, treatment of; soluble receptor BR43x2 isoform
of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating auto
Tumor necrosis factors...
ligand neutrokine α; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating
autoimmune
Antibodies...

monoclonal; soluble receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in modulating
the immune response and treating autoimmune disorders
Nerve,disease...
neuropathy, Ig light chain, treatment of; soluble receptor BR43x2 isoform
of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and trea
Cytokines...
neutrokine α; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune disord
Lymphoma...
non-Hodgkin's, expression specificity in; soluble receptor BR43x2 isoform
of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and trea
Salivary gland...
parotid, neoplasm, expression specificity in; soluble receptor BR43x2
isoform of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and
Animal cell line...
Raji, expression specificity in; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating auto
Shock(circulatory collapse)...
septic, treatment of; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune diso
Intestine...
small, expression specificity in; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating aut
Antibodies... cDNA sequences... Drugs... Gene therapy... Immunosuppression
... Mammal(Mammalia)... Molecular cloning... Mouse... Primate... Protein
sequences...
soluble receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in modulating

the immune response and treating autoimmune disorders
Lupus erythematosus...
systemic, treatment of; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune di
Proteins,specific or class...
TACI (transmembrane activator and CAML-interactor); soluble receptor
BR43x2 isoform of transmembrane activator and CAML-interactor TACI and
related proteins and their use in modulating the immune respons
Amyloidosis... Anemia(disease)... Asthma... Autoimmune disease... Emphysema
... Inflammation... Kidney,disease... Kidney,neoplasm... Multiple myeloma
... Multiple sclerosis... Myasthenia gravis... Rheumatoid arthritis...
Swelling,biological... Transplant rejection...
treatment of; soluble receptor BR43x2 isoform of transmembrane activator
and CAML-interactor TACI and related proteins and their use in
modulating the immune response and treating autoimmune disorders
Fusion proteins(chimeric proteins)...
with Ig heavy chain; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their use
in modulating the immune response and treating autoimmune disor
CAS REGISTRY NUMBERS:
217638-65-8 282738-49-2 amino acid sequence; soluble receptor BR43x2 isoform
of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and treating
autoimmune disorders
283157-99-3 cysteine-rich pseudo repeat domain; soluble receptor BR43x2
isoform of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and treating
autoimmune disorders
156253-82-6 198123-04-5 282738-43-6 282738-45-8 282738-47-0
282738-48-1 nucleotide sequence; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating autoimmune
disorders
148997-64-2D 198029-64-0D 282738-44-7D 282738-46-9D subfragments are

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    claimed, amino acid sequence; soluble receptor BR43x2 isoform of
    transmembrane activator and CAML-interactor TACI and related
proteins
    and their use in modulating the immune response and treating
autoimmune
    disorders
225456-59-7  271755-95-4  274950-83-3  274950-84-4  282738-77-6
    282738-78-7  282738-79-8  282738-80-1  282738-81-2  282738-82-3
    282738-83-4  282738-84-5  282738-85-6  282738-86-7  282738-87-8
    282738-88-9  282738-89-0  282738-90-3  282738-91-4  282738-92-5
    282738-93-6  282738-94-7  282738-95-8  282738-96-9  282738-97-0
    282738-98-1  282738-99-2  282739-00-8  282739-02-0  282739-03-1
    282739-04-2  282739-05-3  282739-06-4  282739-07-5  282739-08-6
    282739-09-7  282739-10-0  282739-11-1  282739-12-2  282739-13-3
    unclaimed nucleotide sequence; soluble receptor BR43x2 isoform of
    transmembrane activator and CAML-interactor TACI and related
proteins
    and their use in modulating the immune response and treating
autoimmune
    disorders
282738-76-5  282739-01-9  unclaimed protein sequence; soluble
receptor BR43x2
    isoform of transmembrane activator and CAML-interactor TACI and
related
    proteins and their use in modulating the immune response and
treating
    autoimmune disorders
98849-88-8  256922-04-0  282729-02-6  282729-03-7  unclaimed
sequence; soluble
    receptor BR43x2 isoform of transmembrane activator and
CAML-interactor
    TACI and related proteins and their use in modulating the immune
    response and treating autoimmune disorders
? ds

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MATURATION AN-
              TIGEN)
S2         17   RD S1  (unique items)
S3       1744   BCMA OR (B(W) CELL(W) MATURATION (W) ANTIGEN)
S4        280   SOLUBLE AND S3
S5         73   RD S4  (unique items)
S6         13   S5 NOT PY>2001
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     $4.53  TELNET
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